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## Iodine-131 and Thyroid Function

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Ostroumova et al. (2013) reported an association between iodine-131 ( $^{131}\text{I}$ ) dose and hypothyroidism in the Belarusian cohort, a cohort of individuals exposed to  $^{131}\text{I}$  from fallout of the Chernobyl accident when they were  $\leq 18$  years of age. Ostroumova et al. also examined other thyroid outcomes: hyperthyroidism, autoimmune thyroiditis, serum concentrations of thyroid-stimulating hormone, and autoantibodies to thyroperoxidase.

It may not be appropriate to include participants with other thyroid outcomes in the analysis because those thyroid outcomes could be indirectly associated with exposure. Chernobyl is in an iodine-deficient area (Ishigaki et al. 2001), and the prevalence of goiters among children  $\leq 18$  years of age has been reported at  $> 15\%$  in this area (Hatch et al. 2011). Is high prevalence of goiters in the area caused by normal iodine deficiency or by the  $^{131}\text{I}$ ? If the goiters were caused by  $^{131}\text{I}$ , the relationship between the  $^{131}\text{I}$  and hypothyroidism is still unclear, even though Ostroumova et al. (2013) stratified the data according to the presence of goiters. Hypothyroidism can also cause goiters (Wilkins et al. 1954); thus, goiter is just a serious hypothyroidism. That could be the explanation for the higher excess odds ratio in the group with goiter compared with the group without goiter shown in Table 3 of Ostroumova et al. (2013). It would have been better for Ostroumova et al. to perform a stratified analysis on the relationship between  $^{131}\text{I}$  and hypothyroidism based on the normal iodine level of the individual rather than the presence of goiter.

Ostroumova et al. (2013) also claimed that the thyroid radioactivity of individuals from the Belarus cohort was based on a previous study (Stezhko et al. 2004). However, Stezhko et al. (2004) did not provide the details of the individual radioactive iodine measurement. Were the original radioactive iodine measurements generated from a formula or modeled based on food intake or soil contamination, or was the  $^{131}\text{I}$  exposure level actually measured for each individual? The answer to this question is necessary because the two methods have different credibility. In addition, the exposure described by Stezhko et al. (2004) included  $^{131}\text{I}$  as well as other radioactive isotopes of iodine, not  $^{131}\text{I}$  alone. I would like to know whether Ostroumova et al. (2013) separated  $^{131}\text{I}$  from other radioactive iodine isotopes. Cesium-137 should also

be considered as a potential confounder in the relationship between  $^{131}\text{I}$  and hypothyroidism.

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## Iodine-131 and Thyroid Function: Ostroumova et al. Respond

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Sun's comments about the relationship between iodine-131 ( $^{131}\text{I}$ ), hypothyroidism, and simple diffuse goiter suggest a misunderstanding of our study findings. We reported a significantly higher—rather than lower—radiation-associated risk of hypothyroidism among study participants without goiter than in the participants with goiter (Ostroumova et al. 2013). Specifically, the excess odds ratio (EOR) per Gray of  $^{131}\text{I}$  thyroid dose was 0.50 [95% confidence interval (CI): 0.24, 0.90] in participants without goiter and 0.04 (95% CI: –0.09, 0.32) in those with goiter. We also reported a lack of significant variation of EOR per Gray for hypothyroidism by levels of urinary iodine ( $p = 0.23$ ), although in the discussion we noted that iodine concentration in spot urine samples, unlike presence of diffuse goiter, reflects current levels of iodine intake and is subject to high within-individual variability.

The territories of Belarus were known to be iodine deficient before the Chernobyl accident;

in the Soviet Union there was a system of iodine prophylaxis that was discontinued by the mid-1980s (Kholodova and Fedorova 1992). In 1995–1998, five of the six Belarus regions were classified as having moderate iodine deficiency, whereas the Gomel region, most heavily contaminated with  $^{131}\text{I}$ , was classified as having mild iodine deficiency partly due to some iodine supplementation in this area after the Chernobyl accident (Arinichin et al. 2000). High prevalence of diffuse goiter detected by ultrasound in children and adolescents in the relatively uncontaminated Brest region (27.8%) and low prevalence in the heavily contaminated Gomel region (5.6%) (Arinichin et al. 2000) support the idea that these differences are attributed to different intake of dietary iodine and not to  $^{131}\text{I}$  exposure. Moreover, there is little evidence of a dose–response association between thyroid exposure and simple diffuse goiter in other radiation-exposed cohorts (Ron and Brenner 2010).

As we described in the “Materials and Methods” of our article (Ostroumova et al. 2013), availability of individual direct measurements of thyroid radioactivity served as a key criterion for inclusion into the study. All study participants had direct measurements of thyroid radioactivity performed within 2 months after the accident. In the methods for dosimetry, we cited the article by Drozdovitch et al. (2013), in which dose reconstruction methods were described in detail. We also noted that intake of  $^{131}\text{I}$  on average accounted for about 95% of the estimated thyroid dose, whereas the contribution of other short-lived radioiodines, external exposures, and internal exposure from cesium-137 and cesium-134 was minor (Bouville et al. 2007).

We appreciate Sun's interest in our study and hope our response is useful.

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